Guidelines for the treatment of abdominal aortic aneurysms

Report of a subcommittee of the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery

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Decision-making in regard to elective repair of abdominal aortic aneurysms (AAA) requires careful assessment of factors that influence rupture risk, operative mortality, and life expectancy. Individualized consideration of these factors in each patient is essential, and the role of patient preference is of increasing importance. It is not possible or appropriate to recommend a single threshold diameter for intervention which can be generalized to all patients. Based upon the best available current evidence, 5.5 cm is the best threshold for repair in an "average" patient. However, subsets of younger, good-risk patients or aneurysms at higher rupture risk may be identified in whom repair at smaller sizes is justified. Conversely, delay in repair until larger diameter may be best for older, higher-risk patients, especially if endovascular repair is not possible. Intervention at diameter <5.5 cm appears indicated in women with AAA.

If a patient has suitable anatomy, endovascular repair may be considered, and it is most advantageous for older, higher-risk patients or patients with a hostile abdomen or other technical factors that may complicate standard open repair. With endovascular repair, perioperative morbidity and recovery time are clearly reduced; however, there is a higher reintervention rate, increased surveillance burden, and a small but ongoing risk of AAA rupture. There is no justification at present for different indications for endovascular repair, such as earlier treatment of smaller AAA. Until long-term outcome of endoluminal repair is better defined and results of randomized trials available, the choice between endovascular and open repair will continue to rely heavily on patient preference. (J Vasc Surg 2003;37:1106-17.)

A decade has elapsed since the Joint Vascular Societies published recommendations on the operative management of abdominal aortic aneurysms (AAA).[1] During this time, much further information regarding the anticipated natural history of unoperated AAA and outcome of conventional open surgical AAA repair has been accumulated.[2] Two carefully performed prospective randomized trials have been published, with findings that challenge many previously held indications for surgical repair.[3,4] Finally, within the past 10 years endovascular AAA repair has evolved and currently plays a major role in AAA management.[5] Thus it is clear that revised guidelines for AAA management are necessary.

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Guidelines are meant to assist physicians in clinical decision making and aim to improve effectiveness of care as well as optimize patient outcomes. In contemporary practice, there is growing emphasis on evidence-based management, and guidelines must therefore be based upon the best available data. It is well recognized that the best evidence (Level I) is derived from properly designed and conducted prospective randomized trials.[6] In regard to AAA management, there are few such trials, and therefore many recommendations are by necessity the result of consensus of participating experts. It should be emphasized that guidelines are not meant to be dictates but rather a framework within which clinicians bring their own judgment in considering unique individual patient circumstances and personal values.

RANDOMIZED TRIALS

Level I evidence for the treatment of small AAA has been provided by two randomized prospective clinical trails conducted in the United Kingdom and the United States.[3,4] Design and results of both trials were remarkably similar. The United Kingdom (UK) Small Aneurysm Trial[3]

and the Aneurysm Detection and Management Study (ADAM),[4] conducted at VA Medical Centers in the US, each examined more than 1000 patients with AAA of 4.0 cm to 5.4 cm in maximal diameter, randomly assigned to early elective open surgical repair or ultrasonographic or computed tomography (CT) surveillance every 3 to 6 months. In the surveillance cohort, operation was recommended if the AAA expanded to ≥5.5 cm, enlarged >1 cm in 1 year, or became symptomatic. The primary endpoint was death, and mortality analyses were done by intention to treat. Mean follow-up was 4.6 years for the UK Small Aneurysm Trial and 4.8 years for the ADAM trial.

Both trials concluded that surveillance of AAA of 4.0 cm to 5.5 cm was safe in compliant patients, and that early surgery did not result in any long-term survival advantage. While operative mortality in the UK trial was higher (5.8%) than had been anticipated, perioperative mortality in the ADAM trial was only 2.7%. Thus, while the UK trial conclusions had been challenged by some because of the relatively high operative mortality, the ADAM trial effectively overcame this concern.

It is important to note, however, that >60% of patients in the surveillance group in *both* studies eventually underwent surgical repair of their AAA because of expansion or development of symptoms by the end of the study. This eventual need for surgical repair was also dependent on the size of the AAA at the time of randomization. In the ADAM trial, for instance, 81% of patients with AAA of 5.0 cm to 5.4 cm at entry into the study required surgical repair within the 4.9-year follow-up period.[4] Rupture risk for AAA in the surveillance group was low in both trials (≤1% per year). One must be cautious in inferring that this figure accurately indicates the natural history of untreated AAA ≤5.5 cm, since 75% of patients in the UK trial, for example, had AAA <5.0 cm, and more than 60% of those in the surveillance group were operated on within the study period following developments considered to signify an increased chance of rupture. Therefore, to interpret these data as accurately reflecting the natural history of AAA up to 5.5 cm is likely somewhat misleading.[7]

In a sequel to their initial report, the UK Small Aneurysm Trial participants have recently described their findings of extended 10-year (1991-2001) follow-up of surviving UK trial patients.[8] By the end of 2001, an additional 12% of the surveillance group had undergone surgical repair, for a total of 74% of patients in this cohort during the 9-year observation of the trial. This emphasizes the fact that for many patients assigned to watchful waiting, the question is often not "if" but rather "when" aneurysm repair will be necessary.[9] In this scenario, patient preferences should be a guiding consideration. These extended trial data revealed worse late survival in the surveillance group; survival curves crossed at about 3 years. At 8 years, the estimated risk of death was 7.2% lower in the early-surgery group than in the surveillance cohort ($P = .03$). However, rupture of unrepaired AAA caused only a small proportion of deaths (6%), so that other explanations must be sought to explain the small late survival advantage in the early surgery group. The trial participants theorized that this difference may be attributable to a higher rate of smoking cessation and other favorable lifestyle changes in the surgery group. An additional important observation was that death was attributable to ruptured AAA in 5% of men who died but 14% of women who died. The risk of rupture was 4 times as high among women as among men. The trial participants concluded that the threshold of 5.5-cm diameter may be too high for women.

INDIVIDUAL DECISION-MAKING

The goal of elective AAA repair is to prevent rupture and prolong life. To be most effective, it should be performed when the rupture risk is high compared with operative risk, in patients who will live long enough to enjoy the long-term benefit. Thus, decision making involved in selecting patients for AAA repair is influenced primarily by estimates of (1) aneurysm rupture risk, (2) elective operative mortality risk, (3) life expectancy, and (4) patient preference. In the absence of truly accurate data regarding many of these variables, decision making is often a complex and uncertain process. It is increasingly recognized that patient preference, after a complete review of options and anticipated results (true informed consent), must be a very important component in this decision-making process.

RUPTURE RISK

Accurate data on rupture risk are likely the least precise of the several variables which need to be assessed in the decision-making process. This is due to the fact that in the past 3 decades few patients have been followed without intervention; hence, the true natural history of untreated AAA remains somewhat poorly defined.[10]

It is accepted that AAA diameter is the best predictor of rupture risk. This was established by natural history studies before the era of widespread elective repair as well as several autopsy studies. The variability of estimates of rupture risk for particular AAA diameters cited in the literature reflects differences in other factors besides maximal diameter which may vary considerably from series to series, and illustrates that other factors in addition to absolute size must be taken into account in each individual case.[11-13]

It is clear that there is a substantial increase in rupture risk as AAA diameter increases from 5 cm to 6 cm. In the only population-based study available, Nevitt et al[14] reported no rupture during 5-year follow-up for AAAs <5 cm, but a 5% annual rupture risk for AAA >5 cm at initial presentation. In a more recent analysis of these data, Reed et al estimated annual rupture risk (with 95% confidence intervals) to be 0% (0%-5%) for AAA <4 cm, 1% (0%-5%) per year for 4.0-4.9 cm AAAs, 11% (1%-21%) per year for 5.0-5.9 cm AAAs and 26% (7%-46%) per year for 6.0-6.9 cm AAAs.[15] Similar estimates were obtained from the larger UK Small Aneurysm Trial, where the annual rupture rate was calculated as 0.3% for AAAs <4 cm diameter, 1.5% for 4.0-4.9 cm AAAs, and 6.5% for 5.0-5.9 cm AAAs.[16] It is possible that these studies underestimate rupture risk since some AAAs underwent elective repair for rapid expan-

Table I. Estimated annual rupture risk

AAA diameter (cm)	*Rupture risk (%/y)*
<4	0
4-5	0.5-5
5-6	3-15
6-7	10-20
7-8	20-40
>8	30-50

sion or symptoms and so were censored before rupture could occur, as previously noted.[7] This issue was considered by Scott et al[17] in analysis of 166 small AAAs with an annual rupture rate of 0.7% for 3.0-4.4 cm AAAs and 1.7% for 4.5-4.9 cm AAAs. Since some AAAs underwent elective repair, they reported maximum possible rupture rates (actual rupture rate plus elective surgery rate) of 2.1% for 3.0-4.4 cm AAAs and 10.2% for 4.5-5.9 cm AAAs.

Although most patients with larger AAAs undergo elective repair, Jones et al[18] reported annual rupture rates of 12% for 5.0-5.9 cm AAAs and 14% for ≥6 cm AAAs in higher-risk or older patients who refused elective repair. Similar striking data relative to rupture risk of large AAA were recently reported by Lederle and colleagues from the ADAM trial data. The 1-year incidence of probable rupture by initial AAA diameter was 9.4% for AAA of 5.5 cm to 5.9 cm, 10.2% for AAA of 6.0 cm to 6.9 cm, and 32.5% for AAA of 7.0 cm or more.[19] Thus, although there is agreement that rupture risk is very low for AAAs <5 cm diameter, and increases substantially by 6-cm diameter, there is considerable variation in estimates of actual rupture risk reported in the literature for any specific AAA diameter (Table I).

The simple observation that not all AAAs rupture at a specific diameter indicates that other patient- or aneurysm-specific variables also affect rupture risk. In a multivariate analysis, Cronenwett et al[20] observed that increased initial diameter, hypertension, and chronic obstructive pulmonary disease (COPD) were independently predictive of rupture in patients with small AAAs. By comparing patients with ruptured and intact AAAs at autopsy, Sterpetti et al[21] concluded that larger initial AAA size, hypertension, and bronchiectasis were independently associated with AAA rupture. Smoking was identified as a risk factor for rupture in a study of male civil servants in England where the relative risk of death from AAA rupture increased 4.6-fold for cigarette smokers, 2.4-fold for cigar smokers and fully 14.6-fold for smokers of hand-rolled cigarettes.[22]

Important new information concerning AAA rupture risk has been obtained from the UK Small Aneurysm Trial data. In a cohort of 2257 patients with 4.0-5.5 cm AAAs, the relative risk of rupture was independently increased by female gender (3.0×), larger initial diameter (2.9× per cm), current smoking (1.5×), worse COPD (0.6× per L FEV1), and higher mean arterial pressure (1.02× per mm Hg).[16] In a review of ruptured AAAs from Finland, 24% of women with rupture had AAA <5.5 cm.[23] These results confirm previous observations and suggest that a 5-cm diameter AAA in a woman has an equivalent risk to a 6-cm diameter AAA in a man.

Not only does a positive family history of AAA increase the prevalence of AAAs in other first-degree relatives (FDRs), but it also appears to increase rupture risk. Darling et al[24] reported that the frequency of ruptured AAAs increased with the number of FDRs who have AAAs: 15% with 2 FDRs, 29% with 3 FDRs, and 36% with 4 FDRs. Verloes et al[25] found that the rupture rate was 32% in patients with familial aneurysms versus 9% in patients with sporadic aneurysms. However, these studies did not consider other potentially confounding factors, such as AAA size, which might have been different in the familial group.

In addition to AAA diameter, many surgeons consider the ratio of diameter to the proximal normal aorta potentially important in determining rupture risk. Intuitively, a 4-cm AAA in a patient with a 1.5-cm diameter native aorta would be at greater risk of rupture than a comparable 4-cm AAA in a patient with a native aortic diameter of 2.5 cm. The validity of this concept, however, has not been proven. Ouriel et al[26] suggested that a relative comparison between aortic diameter and the diameter of the third lumbar vertebra may increase the accuracy for predicting rupture risk, by adjusting for differences in body size. The improvement in prediction accuracy appears minimal, however, when compared with absolute AAA diameter.

Clinical opinion also holds that eccentric or saccular aneurysms represent greater rupture risk than more diffuse, cylindrical aneurysms. Using computer modeling, Vorp et al[27] found that wall stress is substantially increased by an asymmetric bulge in AAAs. In fact, the influence of asymmetry was as important as diameter over the clinically relevant range tested. Fillinger et al have extended this concept to calculate wall stress in AAAs using finite element analysis of three-dimensional CT scans.[28] They found significantly higher wall stress in ruptured or symptomatic AAAs as compared with elective AAAs. In fact, the smallest ruptured aneurysm (4.8-cm diameter) had a calculated wall stress equal to that of a 6.3-cm diameter AAA in the elective repair group. This suggests that calculated wall stress may become a valuable predictor of rupture risk as these techniques become more widely available.

Localized outpouchings or "blebs", ranging from 5 mm to 30 mm in size, can be observed on AAAs intraoperatively or on CT scans. These areas of focal wall weakness demonstrate marked thinning of the medial elastin, and have been suggested to increase rupture risk.[29] Faggioli et al[30] found that impending rupture was significantly greater in patients with such "blisters" than those without (71% vs 29%). The effect of intraluminal thrombus on AAA rupture risk is debated, but a recent study by Schurink et al[31] found that thrombus within an aneurysm does not reduce either mean or pulse pressure near the aneurysm wall and thus does not likely affect rupture risk.

Although rapid AAA expansion is presumed to increase rupture risk, it is difficult to separate this effect from the influence of expansion rate on absolute diameter, which alone could increase rupture risk. AAAs in the 4-cm to 6-cm

Table II. Rupture risk

	Low risk	*Average risk*	*High risk*
Diameter	<5 cm	5-6 cm	>6 cm
Expansion	<0.3 cm/y	0.3-0.6 cm/y	>0.6 cm/y
Smoking/COPD	None, mild	Moderate	Severe/steroids
Family history	No relatives	One relative	Numerous relatives
Hypertension	Normal blood pressure	Controlled	Poorly controlled
Shape	Fusiform	Saccular	Very eccentric
Wall stress	Low (35 N/cm^2)	Mdm. (40 N/cm^2)	High (45 N/cm^2)
Gender	-	Male	Female

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diameter range expand approximately 10% per year.[12,20,32,33] Two studies have reported that expansion rate was greater in ruptured than intact AAAs, but these ruptured AAAs were also larger.[12,34] Even though not proven conclusively, rapid AAA expansion (>1 cm/y) is generally regarded as a risk factor for rupture and is often used as a criterion for elective repair of small AAAs.

Although average AAA expansion rate can be estimated for a large population, it is important to realize that individual AAAs behave in a more erratic fashion. Periods of rapid expansion may be interspersed with periods of slower expansion and are not predictable. Chang et al[35] found that in addition to large initial AAA diameter, rapid expansion is independently associated with advanced age, smoking, severe cardiac disease, and stroke. The influence of smoking has been confirmed by others.[36,37] In addition to these factors, hypertension and pulse pressure have been identified as independent predictors of more rapid expansion rate.[20,33,34] Finally, increased thrombus content within an AAA and the extent of the aneurysm wall in contact with thrombus appear to be associated with more rapid expansion.[38,39]

Although there is no precise formula that incorporates the risk factors described above to calculate exact rupture risk, they can be used to categorize rupture risk as low, average, or high (Table II).

OPERATIVE RISK

As with rupture risk, reported operative mortality of conventional open surgical repair of AAA varies considerably in the literature. Much of this variability is related to the type of study reported, that is, hospital-based versus population-based series.[41] Many referral-based series from individual centers of excellence describe 30-day perioperative mortality of only 1% to 5% following elective open infrarenal AAA repair.[42-44] Such excellent results demonstrate the low mortality rates that can be achieved in selected referral centers by skilled, well-trained, experienced surgeons. However, these data cannot be generalized to larger populations. Thus, it is now well documented that many recent population-based series employing statewide or national databases indicate higher mortality, in the 4% to 8% range even in contemporary practice.[41,45-53] A review of 64 studies on this subject found an average mortality rate of 5.5%.[33] This is consistent with the findings of the UK Small Aneurysm Trial (5.8%),[3] 1996 US Medicare data (5.5%),[2] and the largest available database in the report of Heller et al (5.6%).[47] Results of other population-based studies are similar.[54-60] Surprisingly, there also appears to have been little improvement in mortality rates for elective or ruptured AAA repair over the past two decades.[33,47,49]

Although such generalized experience is important, decision making for individual patients requires a more patient-specific approach. Using individualized estimates of operative risk may clearly identify low- and high-risk subsets of patients and allow more accurate predictions and clinical decisions. Several factors need to be considered. In the Canadian Aneurysm Study, the most significant variables were electrocardiographic (EKG) evidence of ischemia, COPD, and elevated creatinine.[61] If none of these risk factors was present, operative mortality was 1.9%, whereas if all three were noted in a specific patient, 30-day mortality was 50% (Table III, online only). Using the same database from the Canadian Aneurysm Study, an alternative predictive model of operative mortality after AAA repair, which includes the patients' age, has also been developed. Postoperative mortality ranges from 1% to 46% (Table IV, online only). Patient age has also been shown to be an important predictor in the UK study.[62] In this study, the overall postoperative mortality rate was 5.6%. In their subanalsysis, postoperative mortality risk was significantly associated with older age, higher serum creatinine level, and lower forced expiratory volume in 1 second (FEV_1). The predicted postoperative mortality risk ranged from 2.7% in younger patients with normal creatinine levels and good FEV_1 to 7.8% in older patients with elevated creatinine levels and reduced FEV_1. The impact of advancing age has also been shown in many other studies.[53,54]

Similarly, a meta-analysis by Steyerberg et al[63] identified independent risk factors for perioperative mortality of elective open AAA repair (Table V). Based on this analysis, Steyerberg et al also developed a clinical prediction model to estimate the operative mortality risk for individual patients using these factors (Table VI, online only). This scoring system takes into account the independent risk factors plus the average overall elective mortality rate from a specific medical center. Using their scoring system, the predicted operative mortality for a 70-year-old man in a medical center with an average operative mortality rate of 5% could range from 2% if no risk factors were present to

Table V. Independent risk factors for operative mortality after elective abdominal aortic aneurysm repair

Risk factor	*Odds ratio*	*95% CI*
Creatinine >1.8 mg/dL	3.3	1.5-7.5
Congestive heart failure	2.3	1.1-5.2
ECG ischemia	2.2	1.0-5.1
Pulmonary dysfunction	1.9	1.0-3.8
Older age (per decade)	1.5	1.2-1.8
Female gender	1.5	0.7-3.0

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more than 40% if cardiac, renal, and pulmonary comorbidities were all present.

Similar risk factors, as well as the impact of patient-specific variables and the cumulative effect of their influence on operative risk, have also been documented in other reports.[47,52,64,65] In all series, cardiac complications are the predominant cause of perioperative deaths. For example, in Hertzer's report of the Cleveland Vascular Society experience, a mortality rate of 2.9% was observed if patients had a negative history of cardiac disease and normal EKG, 5.2% if either history or EKG was abnormal, and 9.7% if both history and EKG were postive.[55] In the Canadian Aneurysm Study, patients without evidence of coronary artery disease had a 0.8% mortality rate from cardiac disease compared with 6.2% if any stigmata of coronary disease were present.[45]

Additional patient-specific factors need to be considered in estimating perioperative mortality risk in addition to age and the presence of cardiac, renal, or pulmonary comorbidities. Several studies have now documented increased death rates in female patients, with an odds ratio of approximately 1.5 greater risk.[52,56,63,66] It is also strikingly clear that mortality risk is strongly influenced by surgeon training and both surgeon and hospital volume of AAA repair.[2,52,53,57-59,67] For example, analysis of 1996 US Medicare data revealed 30-day operative mortality of 7.9% for low-volume (3 or fewer/y) surgeons as compared with 4.0% for high-volume (11 or more repairs/y) surgeons.[2] In 1996, 60% of surgeons who performed elective AAA repair were low-volume surgeons.[2] Similar findings related to specialty training (vascular surgeons versus general surgeons) and hospital volume have also been observed.[57,59,67] Thus, it is important for a surgeon to know his or her own individual results in assessing risk and making clinical decisions.

Finally, operative mortality risk is influenced by anatomic or pathologic features of an AAA. Such features present technical difficulties and lead to potential complications during graft implantation and hence may impact mortality risk. Extensive atheromatous disease, thrombus formation, or severe mural calcification at sites of proximal or distal anastomosis or clamp application are examples, although actual quantification of the influence of such considerations on the risk of complications or mortality is difficult. Certainly extension of aneurysmal disease to a juxtarenal level, requiring suprarenal clamping for repair, is associated with increased morbidity and mortality due to the more extensive and complex dissection necessary, obligatory renal ischemia time, and increased hemodynamic stresses secondary to more proximal clamping.[68-72] Similarly, inflammatory abdominal aortic aneurysms, with adhesion of adjacent bowel, left renal vein, and/or ureters often present technical challenges and are associated with increased morbidity and mortality.[73,74] A major venous anomaly may occur in as much as 3% to 5% of AAAs and increases the risk of hemorrhage during open surgical repair.[75-77] Thus, mortality may be somewhat higher, particularly when such anomalies are not recognized preoperatively and hence unanticipated during repair.

Again, utilizing an individualized assessment of risk factors for each specific patient allows categorization of operative mortality risk into low (1%-3%), moderate (3%-7%), and high (at least 5%-10% or greater) categories which may be useful on a practical clinical level in terms of decision making (Table VII).

LIFE EXPECTANCY

After estimating rupture risk and anticipated mortality of repair, decision making must also consider the patient's life expectancy. On a population basis, age is the best predictor of life expectancy, which in the United States is approximately 18 years for a 60-year-old man decreasing to 5 years for an 85-year-old man.[78] Obviously, however, for an individual patient, other factors that influence life expectancy must also be considered. Most important, of course, are comorbid medical conditions present in each patient.

In general, 5-year survival following AAA repair is reduced compared to age- and sex-matched population data, averaging approximately 60% to 65% as compared with 75% to 80% anticipated.[79-84] Over the past 2 decades, it appears that the survival rate has not improved significantly, perhaps because selecting higher-risk patients has offset improvements in surgical, medical, and anesthetic management.

As with determination of the risks of AAA rupture and perioperative mortality, individual variables play a considerable role in estimating life expectancy. In the late results of the Canadian Aneurysm Trial, 5-year survival ranged from 27% to 85%.[81] Higher 5-year survival rates were associated with younger age, no history of congestive heart failure, no or minimal angina, no EKG evidence of ischemia, old infarction, evidence of left ventricular hypertrophy or strain or arrhythmia, no significant COPD, and creatinine <1.5. In the large series from the Cleveland Clinic, the predictors of late mortality were age >75 years, a previous history of coronary artery disease (especially with congestive heart failure), chronic pulmonary disease, or creatinine >2.0.[44] Data from Emory University are similar.[85]

Table VII. Operative mortality risk of open AAA repair

Good risk	*Moderate risk*	*High risk*
Age <70 y	Age 70-80 y	Age >80 y
Physically active	Active	Inactive, poor stamina
No clinically overt cardiac disease	Stable coronary disease; remote MI; EF >35%	Significant coronary disease; recent MI; frequent angina; CHF; EF <25%
No other significant comorbidities	Mild COPD	Limiting COPD; dyspnea at rest; O_2 dependency; FEV_1 <1 L/sec
	Creatinine 2.0-3.0	
Normal anatomy	Adverse anatomy or AAA characteristics	Creatinine >3
No adverse AAA characteristics		Liver disease (↑ PT; albumin <2)
Anticipated operative mortality, 1%-3%	Anticipated operative mortality, 3%-7%	Anticipated operative mortality, at least 5%-10%; each comorbid condition adding approximately 3%-5% mortality risk

PATIENT PREFERENCE

Active patient participation in the decision-making process is of paramount importance. This is particularly true for the option of endoluminal repair. Some patients are not psychologically suited to having an untreated AAA with an ill-defined rupture risk. In addition, young patients with AAA in the 4.0-cm to 5.5-cm range will very likely come to eventual repair at some point of follow-up, as demonstrated by several series of selective surgery in which 60% to 75% of patients under surveillance eventually underwent repair.[3,4,20,86] The need for future surgery is also strongly influenced by the size of the AAA at the time of diagnosis. In the original UK Small Aneurysm Trial, 53% of patients with aneurysm 4.5 cm to 4.9 cm at the time of randomization underwent surgical repair within the mean 4.9 years of follow-up, while 81% of those with AAA 5.0 cm to 5.4 cm in diameter came to surgery before conclusion of the trial.[3] Hence, it may be the patient's preference to proceed with repair at a smaller size threshold if operative risk is low. In this regard, follow-up outcomes surveys in the UK Small Aneurysm Trial documented that patients randomized to early surgery had more positive improvements in current health perceptions and health-related quality of life than those patients in the surveillance group.[87] Because the rupture risk is relatively low for AAA <5.5 cm, it should be emphasized that operative results must be outstanding to support early repair. Finally, close patient follow-up achieved in many trials may not be attainable in "real-world" everyday practice. It has been well demonstrated that not all patients will be compliant with the close surveillance necessary in an effective program of watchful waiting.[88] Hence early surgery may be preferable in such patients.

DECISION ANALYSIS MODELS

Because of the complex interaction among variables that influence AAA management, formal decision-analysis models have been constructed to aid in risk comparisons. Such models demonstrate that for a 70-year-old man with average life expectancy and average elective operative mortality (5%), AAA repair will improve life expectancy if annual rupture risk exceeds 1.5%,[89] which is the estimated rupture risk for 4.5-cm to 5.0-cm AAA in many studies. For younger patients, the "threshold" AAA diameter (and rupture risk) that justifies elective repair is lower, whereas in older patients the threshold diameter for elective repair increases.

In a recent decision analysis study employing data from the UK Trial, Schermerhorn and colleagues concluded that early surgery may be cost effective for selected patients with small AAA, particularly younger patients (<72 years of age) with larger AAAs (≥4.5 cm). They emphasize, however, that because the gains in life expectancy are relatively small, clinical decision making should be strongly guided by patient preferences.[90]

ENDOVASCULAR REPAIR

Endovascular aneurysm repair (EVAR) emerged in the early 1990s as an alternative treatment for AAA, and has quickly gained an important role in current clinical management.[5] Many studies have demonstrated equivalent early safety and efficacy of EVAR as compared with conventional open surgical repair.[91-95] In addition, many short-term benefits of EVAR have been documented, including reduced intensive care unit and hospital lengths of stay (LOS), reduced blood loss, fewer major complications, and more rapid recovery.[91-95] Studies with longer follow-up are inconsistent, however; some mid-term reports suggest equivalent outcomes at 3 to 6 years,[96-99] whereas others have raised concerns about the durability of EVAR and highlight the problems of endoleak, need for late reinterventions and/or conversion to open repair, as well as the ultimate failure—rupture.[100-107] At present, there are no randomized prospective clinical trials comparing EVAR with standard open repair, or to continued observation, although several such studies are underway.[108]

Morbidity and mortality. Because of its less invasive nature, most investigators feel that EVAR allows treatment of AAA with lower perioperative mortality risk than conventional open repair in comparable patients. This has not

been conclusively demonstrated, however, as currently available data from Food and Drug Administration trials, as well as registry data and other series, have not shown a statistically significant difference in perioperative mortality. Similarly, no improvement in long-term survival has been reported to date for EVAR as compared with standard open repair.[92,95-97,109,110]

Clearly perioperative morbidity is reduced with EVAR as compared with open operation, with significantly fewer major adverse events. The absolute reduction in complications depends upon the level of stratification of severity, but there are consistent, clinically relevant, relative reductions in complication rates with EVAR, ranging from 30% to 70%. These reductions are primarily in cardiac, pulmonary, and gastrointestinal organ systems. While some earlier studies have found an increased incidence of renal and vascular complications, more recent trials with more experienced operators, smaller delivery systems, and improved device designs have shown no differences in these areas. In addition, EVAR should substantially reduce the incidence of operation-related erectile dysfunction which occurs following standard open AAA repair in a substantial percentage of patients with normal function prior to surgery.[111]

As a consequence of the reduced incidence, as well as severity, of perioperative complications and the less invasive nature of EVAR, recovery time is markedly quicker as compared with conventional open repair.[91,95] Indeed, recovery time from repair is one of the most striking differences between EVAR and open operation for AAA and highlights the sobering review of Williamson et al[112] who observed that up to one third of patients undergoing standard open AAA repair had failed to fully recover at a mean follow-up of 34 months, and 18% of patients stated they would not undergo AAA repair again knowing the recovery process, an outcome that is clearly age-related. Because of the reduced morbidity and quicker recovery associated with EVAR, many authorities believe that this method of treatment is particularly beneficial to older, higher-risk patients who have appropriate anatomy.[113-115]

Endoleak. Mid-term results indicate a generally favorable impact of EVAR on the anticipated natural history of AAA, with limitation of AAA expansion in 80% to 90% of patients and prevention of rupture in 95% to 98%.[5,97-99,116] However, patients must understand potential shortcomings of EVAR that represent a tradeoff for the benefits of less invasive therapy.[5,117] These include persistent or newly-developing late endoleak rates of approximately 10% to 20%.[118]

The true clinical significance of endoleak remains poorly defined, however, and this is indeed a complex and controversial topic.[119-123] Several studies have shown poor correlation between endoleak and outcome, and many authorities believe that the most common variety of endoleak, Type 2 retrograde branch flow, rarely causes adverse clinical consequences.[124-126] In contrast, there is general consensus that Type 1 and 3 leaks are clearly associated with adverse events such as continued AAA enlargement and ongoing rupture risk.[119,126,127] It is also now recognized that aneurysm expansion and even rupture may occur in the absence of a discernible endoleak, a phenomenon which has been termed "endotension."[128]

Secondary interventions. While AAA sac maximal diameter shrinkage was initially received with enthusiasm, longer follow-up has demonstrated that this may be associated with later adverse effects upon the endograft including limb kinkage or occlusion, modular junctional separations, device migration, or related problems.[129-131] Such consequences of late morphologic changes to the AAA have been termed the "paradox of success."[117] Structural deterioration of endoluminal devices appears to increase with time and can also be a source of treatment failure.[98,100] There is some optimism, however, that such problems will be less frequent in the more recently developed second- and third-generation devices.[132,133]

As a consequence of such potentially adverse events following EVAR, it is well recognized that secondary reinterventions are required in as much as 10% of patients per year.[134] The majority of such reinterventions are catheter-based procedures rather than open surgical operations and are generally successful in correcting the problem and maintaining the integrity of the endovascular repair.[98,134,135] Such procedures most often involve stenting for reduced limb flow, coil embolization for endoleaks, or placement of further proximal or distal stent-graft extender components for migration or endoleak. While less invasive and generally successful, the high rate of such secondary interventions contrasts strikingly with conventional open repair in which reintervention rates are less than 2% in the first 5 years. While late reoperation may be required in some patients following open repair, the need for reoperation is generally a late phenomenon and often occurs a decade or more after the initial operative procedure.[136-139] Thus, although the need for catheter-based reintervention does not necessarily indicate failure of EVAR, it is clearly an issue that patients must understand and accept if they elect to undergo endovascular treatment of their AAA.[98]

Conversion to open repair. In initial experience with EVAR, early periprocedural conversion to open operation was necessary in as much as 10% of cases for a variety of technical difficulties or procedural complications.[91,94,98,127] The need for conversion was most often related to poor patient selection, nonflexible large-caliber first-generation devices, and relative operator inexperience.[140] With advancements in all these areas, early conversions are now rare.[5,141,142] Late conversions, however, continue to be required in 1% to 2% of patients per year.[103] Late conversions to open repair are most commonly required for progressive AAA enlargement, device migration, structural failure of the endograft, infection of the prosthesis, and, of course, late AAA rupture. As compared with standard open operative AAA repair, late conversion carries a somewhat higher morbidity and mortality risk due to the frequent need for suprarenal clamping, more extensive dissection, and other potential technical pitfalls, and it is clearly associated with increased risk and worse outcomes.[109,140-142]

AAA rupture. The ultimate failure of endoluminal AAA repair is the occurrence of aneurysm rupture despite a seemingly technically successful endovascular repair. While no ruptures were reported in initial reports of early devices, nearly all devices have, by now, had some aneurysm ruptures after longer follow-up, particularly outside of the controlled circumstances of clinical trials.[105] Analyses of many of these cases have revealed potentially avoidable causes such as poor patient selection, operator deployment errors, or unrecognized/untreated endoleaks.[102,105,143] Aorto-aortic tube endografts and unapproved devices with frequent structural failures have an unacceptably high risk of late rupture and have largely been abandoned. AAA rupture risk in properly selected patients, treated appropriately with approved bifurcated devices, is about 0.5% at 3 to 4 years.[97,98,105,110] The Eurostar Registry experience, which contains a large number of first- and second-generation devices, indicates a cumulative rupture risk of approximately 1% per year.[103] This possibility underscores the importance of continued postprocedural surveillance for patients undergoing EVAR. Patients must understand and accept this more intensive follow-up surveillance burden, potential need for reintervention, and less certain repair of their AAA if they wish to proceed with EVAR.[144]

CHOOSING ENDOVASCULAR VERSUS OPEN AAA REPAIR

The availability of EVAR has added additional considerations and complexity to surgical decision making for AAA repair. With the lower mortality and morbidity of EVAR, this approach could be assumed to justify repair of smaller aneurysms if they have favorable anatomy. However, at present the late complications and higher reintervention rate offsets this potential advantage, and indications for repair should remain the same.[145] A recent decision analysis by Schermerhorn et al[146] using Eurostar data for endovascular repair, and Medicare data for open repair, found little difference in quality-adjusted life expectancy between the two strategies. Although EVAR was slightly more beneficial (except in younger patients), small variations in many key variables changed the optimal strategy for any given patient.

Thus, decisions regarding the optimal method of AAA repair in an individual patient will remain uncertain until long-term outcomes of EVAR are more clearly established. These data will be best obtained from large prospective registries employing prospective pooled data and continued careful scientific analysis.[147] Randomized control trials comparing EVAR with standard open operation or best medical therapy in very high-risk patients will hopefully provide more definitive information and thereby facilitate the decision-making process. For now, the choice between EVAR and conventional open surgery will continue to rely heavily on patient preference. This is valid, however, only if patients are fully informed and aware of potential benefits as well as disadvantages of both methods. This remains an important responsibility of the treating surgeon.

CURRENT RECOMMENDATIONS FOR AAA REPAIR

1. The arbitrary setting of a single threshold diameter for elective AAA repair applicable to all patients is not appropriate, as the decision for repair must be individualized in each case.
2. Randomized trials have shown that the risk of rupture of small (<5 cm) AAA is quite low, and that a policy of careful surveillance up to a diameter of 5.5 cm is safe, unless rapid expansion (>1 cm/y) or symptoms develop. However, early surgery is comparable to surveillance with later surgery, so that patient preference is important, especially for AAA 4.5 cm to 5.5 cm in diameter.
3. Based upon the best available current evidence, 5.5-cm diameter appears to be an appropriate threshold for repair in an "average" patient. However, subsets of younger low-risk patients, with long projected life-expectancy, may prefer early repair. If the surgeon's personal documented operative mortality rate is low, repair may be indicated at smaller sizes (4.5-5.5 cm) if that is the patient's preference.
4. For women, or AAA with greater than average rupture risk, elective repair at 4.5 cm to 5.0 cm is an appropriate threshold for repair.
5. For high-risk patients, delay in repair until larger diameter is warranted, especially if EVAR is not possible.
6. In view of its uncertain long-term durability and effectiveness, as well as the increased surveillance burden, EVAR is most appropriate for patients at increased risk for conventional open aneurysm repair.
7. EVAR may be the preferred treatment method for older, high-risk patients, those with "hostile" abdomens, or other clinical circumstances likely to increase the risk of conventional open repair, if their anatomy is appropriate.
8. Use of EVAR in patients with unsuitable anatomy markedly increases the risk of adverse outcomes, need for conversion to open repair, or AAA rupture.
9. At present, there does not appear to be any justification that EVAR should change the accepted size thresholds for intervention in most patients.
10. In choosing between open repair and EVAR, patient preference is of great importance. It is essential that the patients be well informed to make such choices.

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